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WALTER M. DICKIE, M.D., Director

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Epidemic Meningitis[*]

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The incidence curve of epidemic meningitis in California by years and months from 1920 to the end of July, 1929, shows that there was relatively a high incidence of the disease in 1920 and 1921, but thereafter, for a period of four years, there were few cases and few deaths. In December, 1925, there was a definite rise in incidence which marked the beginning of a series of three annual peaks which occurred in the winter and spring months of 1926, 1927 and 1928. The peak of 1928 was lower than those of 1926 and 1927, and during the late summer and autumn months of 1928 the incidence curve appeared to be approaching the normal level of 1922–1925. However, in November, 1928, the case incidence began to increase and continued to rise rapidly, month by month, until the peak of 112 cases was reached in March, 1929. A high incidence was maintained during April and May, but in June and July the number of new cases had dropped to about one-half, although this still was relatively a high case incidence. There has been a further decline during the past few weeks, but, bearing in mind that the normal seasonal incidence is greater in the winter and spring months, one can but speculate whether we have passed through the worst of this epidemic or whether there is more to follow.

An interesting, though not surprising fact is that, coincident with the marked increase in the number of cases there has been an increase in the case mortality rate. From 1922 to 1925, inclusive, the case mortality rate was 36.6 per cent; in 1928 it was 44.4 per cent; and during the first six months of 1929 it was 50.8 per cent. The number of reported cases during this six-month period was 521, and the number of deaths was 265.

An editorial in the Journal of the American Medical Association of June 15, 1929, might lead one to believe that the epidemic meningitis situation on the Pacific coast was particularly alarming, but no such conclusion is justifiable. This has been a meningitis year, not only in the West but elsewhere in the United States, and the situation has been sufficiently acute to justify monthly bulletins in the Public Health Reports. On January 11 it was reported that, although the incidence of epidemic meningitis in the United States had been unusually low during the first week in November, 1928, a sharp rise became apparent during the remainder of the month and that during the three weeks ending December 1, 258 cases had been reported as compared with 139 cases during the corresponding period of 1927. On February 1 it was reported that there was an increase during December in nearly all the states, and on March 1 it was stated that in January, 1929, the incidence of the disease in the United States was the highest since 1918, with a general tendency towards an increase in all sections of the country. Finally, on July 5, 1929, McCoy reported that one must go back to 1905 to find the last comparable prevalence of the disease, and it is an interesting fact that the beginning of the epidemic was at approximately the same time in all sections of the country.

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[*] Read before Health Officers' Section, League of California Municipalities, Oakland, Oct. 10, 1929.

Another point of interest in the epidemic in California is the relatively high incidence of a virulent form of the disease among Filipinos and Chinese who were brought to the United States by various steamship lines. The route of the vessels which brought in patients was from Manila, via various Oriental ports to Honolulu, and thence to the Pacific coast ports. According to the Journal of the American Medical Association of June 15, 128 cases of meingococcus meningitis occurred on fourteen ships of one transportation company, many of the victims dying at sea. All of the patients were steerage passengers or members of the crew who had intimate contact with them. There were no cases among the cabin passengers.

There is still some doubt as to the origin of the infection of these steerage passengers, but all investigators are agreed that the crowded condition and lack of ventilation in the steerage quarters of the ships were responsible for the rapid spread of the infection once it had been introduced. Active measures were taken for the control of the situation. On June 21, President Hoover issued an executive order restricting, for the time being, the transportation of passengers from certain ports in the Orient to a United States port. By this order all immigration from China and the Philippines was placed under the direct control of the United States Public Health Service, the number of steerage passengers permitted to embark was reduced to 25 per cent of the number formerly permitted (this was increased later to 50 per cent under certain controlled conditions), and strict regulations were outlined as to what precautions should be taken at the port of embarkation, during passage to this country, and at the port of landing in this country. The full text of this order has been published in the State Department of Health Weekly Bulletin of August 10.

There is an important problem arising from the immigration of these infected Orientals. The entrance of persons who are actually ill with the disease is of economic importance because they must be cared for, but they do not constitute an important factor in the spread of the disease in this country because they are isolated as soon as they are brought ashore. The admission of carriers, however, is another matter. Should they escape recognition and be permitted to go their way, there is possibility that they may spread the disease among susceptible individuals with whom they come in contact. Murray of the Medical Research Council of Great Britain (Special Report, Series No. 124) states that 95 per cent of all cases of epidemic meningitis are infected by contact with healthy carriers, and when, as is the case with the Filipinos, the carriers live in crowded quarters with many other people under poor hygienic conditions, the opportunity for rapid dissemination of the disease is very great. It was on this account that the immigrant Filipinos were undoubtedly a factor in the spread of the disease in San Francisco and in Monterey County.

But, not only may immigrant carriers spread the disease among our susceptibles, but there is possibility that they may introduce new strains of the meningococcus, strains against which our therapeutic anti-sera are inert. Many strains of the meningococcus have been identified, and it is on this account that commercial anti-meningococcus therapeutic sera are polyvalent. Rosenau (Preventive Medicine and Hygiene, 5th edition, p. 255) states that the Hygienic Laboratory recognizes twelve strains of meningococcus, four agglutinating and five tropin groups, but there may be others. Although it is true that the agglutinability of a strain by an antiserum is not a complete index of the therapeutic value of the antiserum in the treatment of a patient who is infected with that particular strain, it is generally believed that a serum which agglutinates the strain is more potent. Indeed, it is recommended by some authorities that, whenever possible, the strain which is responsible for the infection of a patient should be isolated and tested for type by agglutination tests, and that specific monovalent serum for that type should be substituted for polyvalent serum in the treatment, always, however, using the polyvalent serum until the identification is established.

There is no available information as to whether the strains of meningococcus which were introduced by the immigrant Filipinos were different from those which have been recognized in this country. It is a fact that bacteria were isolated from a number of patients during the past months, including some Filipinos, which showed very poor agglutination by available anti-sera, but similar observations have been made in other parts of the country. Pope and White of Chicago (Jour. Prev. Med., 1929, 3:63) showed that several different strains of meningococcus may be active in a single epidemic. Among nine coincident cases in that city they found three patients with Type I, two with Type III and four with Type IV meningococcus, although in no instance was there any indication from the clinical course or the epidemiology as to which strain was responsible.

The case mortality rate in California during the past few months was, as has been stated, unfortunately high, 50.8 per cent of reported cases, but we can not assume that this was entirely due to treatment with nonspecific antimeningococcus serum. A number of factors must be considered before we can arrive at any definite conclusion as to the efficacy of our serum theapy. It is true of specific anti-sera of all kinds that they are of maximum efficiency in treatment only when they are administered early in the course of the disease, and that their potency diminishes in proportion to the length of time which elapses after the onset of the disease before they are administered. In judging the value of our serum therapy, therefore, it is only fair to eliminate, or at least to segregate in our records, those fulminant cases in which death occurs very early, and all cases in which the serum was first administered several days after the onset of illness. In fact, to obtain a true picture of the value of serum therapy, one should tabulate together all cases in which the serum was first administered on corresponding days of the disease, and, of course, the amount of serum given and the make should be recorded.

It is unfortunately true that all available anti-meningococcus serum is not of equal potency in a given epidemic. Pope and White (loc. cit.) report that among nineteen patients who were treated with a serum which they designate as I, the death rate was 79 per cent; among twenty-five treated with serum II it was 44 per cent; and among thirty-nine who were treated with serum V it was only 21 per cent; whereas, six patients who were untreated with serum all died. Their investigation was undertaken because the case mortality rate in Chicago, like that in California, was high—46.4 per cent.

But Pope and White also found that other factors were of apparent importance in producing this high death rate. The majority of their 194 cases were treated in two hospitals, and in these the death rate was much lower than in those hospitals where only a few patients were admitted. They attribute this to the greater experience and better facilities for dealing with the diagnosis and treatment of the disease in hospitals where many cases are treated. Moreover, they found that the mortality was lowest when the serum was administered within twenty-four hours after the onset of illness, only slightly higher if treatment was begun within three days, but that it rapidly increased if the beginning of treatment was longer delayed. Their investigations established once more that early diagnosis and early administration of antiserum in sufficient quantities are the most important factors in the successful treatment of this disease.

From an administrative standpoint, it is probable that the most difficult problem in the control of epidemic meningitis is the control of carriers. They are of two kinds, primary and secondary or convalescent, and both kinds are potentially infective.

Secondary or convalescent carriers are persons who, during and after convalescence from the disease, continue to harbor virulent meningococci in the naso-pharynx. All victims carry these bacteria at some time during their illness, but the majority are free within fourteen days from the onset of illness, and it is on this account that the period of isolation is usually set at two weeks. It must be remembered, however, that a few patients do not become bacteria free within two weeks, but become chronic carriers, and that they can only be recognized by bacteriological tests.

Primary carriers are healthy persons who have not suffered from the disease but who harbor meningococci in the naso-pharynx. They may be classed as contact carriers if they have been associated with patients who have meningitis, and as noncontact carriers if no such contact can be traced. The noncontact carriers are, perhaps, the greater menace because no one suspects that they are carriers.

The contact carrier rate is usually higher than the noncontact rate, especially in time of epidemic, a fact that has recently been demonstrated in Detroit (Journal Amer. Med. Assn. 1929, 92:1456, Medical News). In a general survey of unexposed persons only 6 per cent were found to be carriers of meningococci, whereas among 803 household contacts of patients with meningitis, the carrier rate was 46.2 per cent. Nevertheless, should a carrier of any type be introduced into an environment which is suitable for the spread of the disease, an environment where there is overcrowding, poor ventilation, poor hygienic arrangements, etc., the carrier rate increases rapidly and cases of the disease may appear. This is obviously what happened on the Pacific steamships.

Glover (cited from Murray, loc. cit.) has pointed out that when the noncontact carrier rate reaches 20 per cent it is a sure storm signal, and that any increase should be considered as a warning. However, cases may occur when the carrier rate is low, especially if carriers and susceptible individuals come together under conditions favorable for spread, since the susceptible individuals may either contract the disease or become carriers. One can not, therefore, depend too much upon the carrier rate as an index of approaching epidemic, even if it is possible to estimate their number.

Primary carriers, as well as secondary, are usually only temporarily infected. McCoy (loc. cit.) says that most carriers clear up within a few days regardless of what one does or does not do, and Murray states that 90 per cent of primary carriers are free from infection within six weeks, not more than 2 per cent continuing to be carriers for from six to twelve months. The consensus of opinion is that there is no satisfactory drug therapy for meningococcus carriers, and anti-meningococcus anti-serum does not help. Fresh air and sunshine seem to clear them up promptly if sleeping quarters are not crowded and are well ventilated. It is impossible to say what proportion of the Filipinos were carriers when they were quarantined in supervised camps in Monterey County, but it is remarkable that after the period of quarantine had elapsed, not a single carrier was found among 423 persons who were examined by Miss Beck, Assistant Epidemiologist of the State Department of Health.

MORBIDITY*

Diphtheria.

55 cases of diphtheria have been reported, as follows: Berkeley 3, Oakland 1, Fresno County 1, Los Angeles County 2, Alhambra 1, Glendale 5, Long Beach 1, Los Angeles 16, Whittier 1, Mendocino County 1, Merced 1, Riverside County 2, San Francisco 9, San Joaquin County 1, Stockton 3, Paso Robles 2, San Jose 2, Tuolumne County 1, Oxnard 2.

Scarlet Fever.

200 cases of scarlet fever have been reported, as follows: Berkeley 1, Oakland 11, San Leandro 5, Colusa County 1, Contra Costa County 1, Fresno County 25, Fresno 7, Humboldt County 1, Imperial County 2, Kern County 2, Bakersfield 2, Corcoran 1, Los Angeles County 2, Arcadia 1, Burbank 1, Culver City 2, Glendale 2, Inglewood 2, Long Beach 2, Los Angeles 40, Whittier 1, Torrance 1, Madera County 1, Marin County 6, Salinas 4, Anaheim 1, Fullerton 1, Riverside 1, Sacramento 10, San Diego 1, San Francisco 5, San Joaquin County 3, Lodi 5, Stockton 4, San Luis Obispo County 2, Arroyo Grande 1, San Mateo County 1, Burlingame 1, South San Francisco 1, Menlo Park 3, Santa Clara County 2, San Jose 4, Siskiyou County 12, Dunsmuir 4, Mt. Shasta 4, Rio Vista 1, Vallejo 2, Sonoma County 3, Tulare County 3, Tuolumne County 1.

Measles.

59 cases of measles have been reported, as follows: Oakland 2, Inyo County 1, Los Angeles County 2, Burbank 2, Inglewood 1, Long Beach 1, Los Angeles 4, Monrovia 1, Santa Monica 1, Maywood 1, Sacramento 1, San Francisco 18, Daly City 20, Menlo Park 1, Santa Barbara County 1, Santa Cruz County 1, Santa Paula 1.

Smallpox.

17 cases of smallpox have been reported, as follows: Oakland 2, Los Angeles County 1, Pomona 1, Salinas 1, Nevada County 2, Grass Valley 2, San Diego 1, San Francisco 1, San Luis Obispo County 1, San Luis Obispo 1, Santa Clara 2, Stanislaus County 1, Yuba County 1.

Typhoid Fever.

13 cases of typhoid fever have been reported, as follows: Imperial County 2, Los Angeles County 2, Los Angeles 1, Whittier 3, Chowchilla 1, Riverside County 1, San Bernardino County 1, San Joaquin County 1, San Jose 1.

Whooping Cough.

76 cases of whooping cough have been reported, as follows: Berkeley 2, Oakland 10, Bakersfield 1, Los Angeles County 3, Culver City 1, Huntington Park 1, Los Angeles 19, Pasadena 1, Orange County 8, Anaheim 3, Huntington Beach 5, Santa Ana 4, Riverside County 1, Sacramento 2, San Diego 6, San Francisco 4, Lodi 1, Stockton 1, Benicia 1, Exeter 1, Tuolumne County 1.

Meningitis (Epidemic).

3 cases of epidemic meningitis have been reported, as follows: Los Angeles County 1, San Diego 1, Daly City 1.

Poliomyelitis.

Colusa County reported one case of poliomyelitis.

Food Poisoning.

3 cases of food poisoning have been reported, as follows: Covina 1, Los Angeles 2.

Undulant Fever.

Merced County reported one case of undulant fever.

Coccidioidal Granuloma.

San Jose reported one case of coccidioidal granuloma.

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* From reports received November 4th and 5th for the week ending November 2d.

COMMUNICABLE DISEASE REPORTS

Disease	1929				1928			
	Week ending			Reports for week ending Nov. 2 received by Nov. 5	Week ending			Reports for week ending Nov. 3 received by Nov. 7
	Oct. 12	Oct. 19	Oct. 26		Oct. 13	Oct. 20	Oct. 27	
Actinomycosis	0	0	0	0	0	1	0	0
Anthrax	0	0	1	0	0	0	1	0
Chickenpox	102	205	180	179	80	169	154	196
Coccidioidal Granuloma	1	2	0	1	1	0	0	0
Diphtheria	56	68	79	55	67	90	103	103
Dysentery (Amoebic)	0	1	1	0	0	0	1	0
Dysentery (Bacillary)	1	1	2	1	0	1	2	1
Encephalitis (Epidemic)	1	0	0	1	0	5	0	1
Erysipelas	9	14	8	13	9	9	14	0
Food Poisoning	1	2	0	3	0	13	3	0
German Measles	8	10	12	5	4	10	15	10
Gonococcus Infection	129	146	132	103	80	90	121	104
Hookworm	0	1	0	0	0	0	0	0
Influenza	30	26	32	24	44	171	1,557	2,389
Leprosy	0	1	0	1	1	0	0	0
Malaria	2	2	5	0	1	1	5	2
Measles	44	54	42	59	12	27	15	15
Meningitis (Epidemic)	7	10	6	3	3	0	5	3
Mumps	201	186	289	244	87	200	154	213
Ophthalmia Neonatorum	0	0	1	1	0	0	0	0
Paratyphoid Fever	1	0	0	1	0	1	1	0
Pellagra	1	2	0	4	1	1	0	0
Pneumonia (Lobar)	45	43	28	25	28	34	60	51
Poliomyelitis	5	5	0	1	2	1	7	4
Rabies (Animal)	16	18	15	5	13	12	14	8
Scarlet Fever	148	157	151	200	92	140	184	179
Smallpox	22	24	37	17	31	27	17	6
Syphilis	145	131	170	256	151	121	229	130
Tetanus	1	1	0	0	2	0	1	2
Trachoma	6	1	0	0	160	3	3	7
Tularemia	0	0	0	0	0	0	1	0
Tuberculosis	199	218	181	171	185	208	264	216
Typhoid Fever	18	12	10	13	17	15	15	11
Undulant Fever	4	2	2	1	0	0	0	1
Whooping cough	112	109	72	76	124	169	170	179
Totals	1,315	1,452	1,456	1,463	1,195	1,519	3,116	3,831

Whooping cough has diminished greatly in its prevalence.

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Scarlet fever shows a marked increase.

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Diphtheria continues at a low level.

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Mumps is quite prevalent.

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